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Integrated control of potato late blight: predicting the combined efficacy of host resistance and fungicides

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Integrated control of potato late blight: predicting the combined efficacy of host resistance and fungicides

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2

3 **Integrated control of potato late blight: predicting the combined efficacy of host**
4 **resistance and fungicides**

5

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16

17

18 **Abstract**

19 Integrating cultivars that are partially resistant with reduced fungicide doses offers growers
20 an opportunity to decrease fungicide input but still maintain disease control. To use integrated
21 control strategies in practice requires a method to determine the combined effectiveness of
22 particular cultivar and fungicide dose combinations. Simple models, such as additive dose
23 models (ADM) and multiplicative survival models (MSM), have been used previously to
24 determine the joint action of two or more pesticides. This study tests whether a model based
25 on multiplicative survival principles can predict the joint action of fungicide doses combined
26 with varieties of differing partial host resistance. Data from eight field experiments on potato
27 late blight (*Phytophthora infestans*), where the severity of foliar blight was assessed and
28 converted to AUDPC, were used to test the model. A subset of data, derived from the most
29 susceptible cultivar, King Edward, was used to produce dose response curves from which
30 parameter values were estimated, quantifying fungicide efficacy. These values, along with the
31 untreated values for the more resistant cultivars, Cara and Sarpo Mira, were used to predict
32 the combined efficacy of the remaining cultivar by fungicide dose combinations. Predicted
33 efficacy was compared against observations from an independent sub-set of treatments from
34 the field experiments. The analysis demonstrated that multiplicative survival principles can
35 be applied to describe the joint efficacy of host resistance and fungicide dose combinations.

36 Introduction

37 There are many ways in which integrated control strategies for foliar plant pathogens can be
38 deployed. Examples of using host resistance to limit damage or reduce dependence on
39 fungicides include switching from susceptible to moderately resistant varieties for control of
40 northern corn leaf blight of maize (*Exserohilum turicum*) and decreasing the number of
41 fungicide applications on wheat varieties with resistance to tan spot (*Drechslera tritici-*
42 *repentis*) (Debela *et al.*, 2017, Jørgensen & Olsen, 2007). Decreasing fungicide inputs on
43 moderately resistant potato cultivars compared with susceptible cultivars has been shown to
44 be an effective control strategy against late blight, caused by *Phytophthora infestans* (Fry,
45 1978, Gans *et al.*, 1995, Nærstad *et al.*, 2007). Despite considerable research demonstrating
46 the potential to optimise inputs by combining strategies, e.g. fungicide inputs and genetic
47 resistance of varieties, no study has yet determined whether there is a predictable relationship
48 describing the joint action of different components of integrated control.

49 Simple models to predict the joint action of two or more pesticides applied in mixture
50 have been used in laboratory and field studies for invertebrate pests, weeds and diseases
51 (Bliss, 1939, Scardavi, 1966, Colby, 1967, Rummens, 1975, Gisi *et al.*, 1985, Paveley *et al.*,
52 2003). In these simple models, the efficacy of each component of the mixture is quantified
53 and used to predict the efficacy of the mixture. The efficacy of treatment combinations has
54 been described as synergistic or antagonistic where the joint action of the mixture
55 components exceeded or failed to achieve the level of control predicted. More recently, one
56 such model has been applied to assess the joint action of host resistance genes (identified as
57 quantitative trait loci) against diseases of winter wheat (Grimmer *et al.*, 2015).

58 The two most frequently used models to determine the combined efficacy of two or
59 more control methods have been defined as two broad types, additive dose models (ADM)

60 and the multiplicative survival models (MSM), also known as the Abbot and Wadley
61 methods respectively. Their respective appropriateness for joint action comparisons has been
62 reviewed previously (Morse, 1978). The ADM assumes that the action of one component can
63 be directly substituted for the action of the other, i.e. that the dose of one mixture component
64 can be expressed as an equivalent dose of the other component. It has been reported
65 previously that the effects of host resistance and fungicides to control *Phytophthora infestans*
66 were 'additive' (Fry, 1978). However, the assumption underlying the ADM cannot be met
67 when the two components have fundamentally different modes of action, such as in the case
68 described here where host resistance and fungicides are combined. MSM, however, calculates
69 the proportion of the pathogen population which 'survives' the effect of each component
70 separately, and then predicts the proportion of the population which would survive joint use
71 of the control methods by multiplying the survivorship proportions. The underlying
72 assumption is that the two components act independently, which is a plausible assumption for
73 host resistance and fungicides. Hence, MSM is likely to be the more appropriate model for
74 determining the joint action of host resistance and fungicide dose.

75 This study tested whether a simple multiplicative survival model can predict the joint action
76 of fungicide dose and host resistance against *Phytophthora infestans* on potato and could
77 provide an accurate prediction of the performance of those combinations under field
78 conditions.

79

80 **Materials and methods**

81 Data derived from eight integrated control field experiments conducted in 2010 and 2011
82 were used in this study. The experiments were conducted on late blight (*Phytophthora*
83 *infestans*) of potato (*Solanum tuberosum*) at two sites: Ayrshire and Ceredigion in the UK.

84 Two experiments were conducted at each site in each year: one with treatments applied
85 during rapid canopy growth ('rapid canopy') and the other with treatments applied later in the
86 seasons when canopy size was relatively stable ('stable canopy').

87

88 *Experimental design*

89 Treatments in each experiment consisted of all combinations of three cultivars and four
90 fungicide doses, with an untreated control for each cultivar (Table 1). Integrated control
91 treatments were considered to be the treatments where cultivars, which were moderately
92 (Cara) or highly (Sarpò Mira) resistant, were combined with fungicide doses below the
93 maximum permitted dose per application (the full label recommended dose). At both sites
94 experiments were laid out as a randomised split-plot design with four replicates for each
95 treatment. Fungicides were applied at the whole plot level and cultivars planted at the subplot
96 level. Fungicides were randomised within each block and varieties randomised within each
97 fungicide plot. In Ceredigion, main plots were four rows wide (each row = 0.9m wide) by
98 11m long. Main plots were separated by 1.5 m unplanted row length. Each main plot was
99 divided into cultivar sub-plots of four rows by 3.0m long and separated by 1.0m unplanted
100 row length. All cultivars were supplied as the same seed size (35 to 45mm) and planted at
101 30cm spacing. A single row of King Edward was planted between each of the blocks as an
102 infector row.

103 In Ayrshire in 2010, fungicide treatment plots were four rows wide by 9.75 m long
104 and separated longitudinally by 1.5 m of bare earth. Each cultivar sub-plot was four rows by
105 2.75 m long separated by 0.75 m unplanted row length with 25cm seed spacing. In 2011, seed
106 spacing was 0.23 m. The fungicide treatment plots were 8.97 m long and the cultivar plots 2.3

m long. The unplanted row length was 1.61 m. A single row of King Edward was planted longitudinally between each of the blocks as an infector row.

One fungicide, mandipropamid [full recommended label rate 0.6 l ha^{-1} (250 g l^{-1}) as Revus, Syngenta Ltd] was applied to cultivars at a range of doses, as proportions of the full recommended label rate (Table 1). Treatments were applied either during rapid canopy and or during stable canopy growth in separate experiments. For the rapid canopy experiments, first treatment fungicides were applied at the time of the first blight warning or when plants met within the rows, whichever was soonest. For the stable canopy experiments, chlorothalonil + propamocarb-hydrochloride [full recommended rate 2.5 l ha^{-1} ($375 \text{ g l}^{-1} + 375 \text{ g l}^{-1}$) as Merlin, Bayer CropScience] were applied to all plots at 7- or 10-day intervals until rapid canopy growth was complete (typically three applications at 10-day intervals per season). For all experiments, four fungicide applications for each treatment at 7-day intervals were planned, however, there was flexibility depending on the epidemic progress at different sites and for different seasons (Table 5). In Ceredigion, there were four applications of treatment fungicides in app experiments conducted in 2010 and 2011. In Ayrshire, there were four test fungicide applications in the 2010 rapid canopy experiment and five in the equivalent trial in 2011. Both stable canopy trials had six applications of test fungicides. Once treatment sprays had been applied, all plots were sprayed with between 1274 g ha^{-1} and 1540 g ha^{-1} mancozeb (as Dithane NT, Penncozeb or Laminator Flo depending on site and season) at 7-day intervals until desiccation. In Ceredigion, fungicide treatments were applied using a handheld Oxford Precision Sprayer in 250 litres of water per hectare operating at 200 kPa through 110° flat fan nozzles. In Ayrshire, treatments were applied in 200 litres of water per hectare, using a tractor-mounted modified AZO compressed air sprayer through Lurmark F03-110 flat fan nozzles at an operating pressure of 350 kPa.

131 Infector rows (cv. King Edward) were not sprayed with fungicide and were inoculated
 132 with isolate(s) of genotype 13_A2 grown on Rye B agar (Caten & Jinks, 1968) but sub-
 133 cultured several times on detached King Edward potato leaves prior to inoculation of
 134 experiments. These isolates were used to produce a sporangial suspension (a minimum of $1 \times$
 135 10^4 spores ml^{-1}) in sterile distilled water which was applied to the spreader rows using a
 136 handheld mister. The isolates were supplied by the James Hutton Institute, Dundee, UK
 137 (Table 2).

138 Foliar blight was assessed at least weekly as the percentage of leaf area affected by *P.*
 139 *infestans*, with more frequent assessments when the epidemic was increasing rapidly, using
 140 the modified MAFF key 2.1.1: Potato Blight on the Haulm (Anon, 1976; Large, 1952).

141

142 *Determining the effectiveness of cultivar and fungicide dose combinations*

143 For each treatment the Area Under the Disease Progress Curve (AUDPC) was calculated
 144 from the foliar late blight severity scores (Campbell & Madden, 1990). To predict the
 145 effectiveness of host resistance and fungicide combinations, a multiplicative survival
 146 equation was derived incorporating a previously published exponential equation (1)
 147 describing the fungicide dose response curve, where D_d is disease severity at dose d and D_o is
 148 disease when fungicide dose = 0 (Paveley *et al.*, 2000).

$$149 \quad (1) \quad D_d = D_o[1 - b(1 - e^{-kdose})]$$

150

151 Data generated from the observed dose response curve of the most susceptible cultivar, King
 152 Edward, were used to calculate the parameters b and k , where b represents the amount of
 153 disease that might be potentially controlled with an infinite dose and k defines the rate of
 154 change of disease severity with dose. Curves were forced through the untreated values and

parameters calculated using the following equation in FITNONLINEAR in Genstat 16th edition (VSN International Ltd, UK).

157

The dose response equation and parameters were then used in the following equation (2) to predict the effectiveness of cultivar and fungicide dose combinations based on the principles of multiplicative survival:

$$(2) \quad D = D_o \left[\left(\frac{D_r}{D_s} \right) (1 - b(1 - e^{-kdose})) \right]$$

D is the predicted level of disease for the appropriate cultivar and fungicide dose combination, D_o is the untreated AUDPC of the standard susceptible cultivar (in this case the most susceptible cultivar King Edward). For the first analysis, D_s is the untreated AUDPC for the standard cultivar, D_r is the untreated AUDPC for the partially resistant test cultivar and $dose$ is the proportion of the full fungicide dose.

AUDPC values were logit transformed using an equation (3) modified from Grimmer *et al.* (2015):

$$(3) \quad LTS = \ln[s/(M - s)]$$

Where LTS is the logit transformed AUDPC, \ln is the natural logarithm, s is the observed or predicted disease severity and M is the maximum AUDPC achievable during the disease assessment period (e.g. if disease assessment period was 59 days then maximum AUDPC is 5900). The transformed observed severity was linearly regressed against the transformed predicted severity as advocated in Piñeiro *et al.* (2008). All analysis was done in Genstat 16th Edition (VSN International Ltd, UK).

Results

Parameter estimates for the dose response curves were derived from dose response curves generated using King Edward as the baseline. The fitted dose response curves were close to the observed AUDPC values (Figure 1). The percentage variance accounted for (as R^2) for all fitted curves ranged from 96% to 100% (Table 3). Observed and predicted disease severities, as the AUDPC for each cultivar and fungicide dose combination, for moderately resistant Cara and highly resistant Sarpo Mira cultivars were compared (Table 4). There was a highly significant relationship ($P < 0.001$; $R^2 = 0.88$) between the predicted and observed values for each fungicide dose and cultivar combination (Figure 2). When the combined effects of fungicide dose and cultivar were predicted, the resultant AUDPC values were generally higher than those observed in the experiments, regardless of cultivar resistance rating.

The percentage of foliar late blight present at the first and last fungicide application in each trial varied depending on the site and year (Table 5). In 2010, only the stable canopy trial in Ayrshire had foliar late blight present when first fungicides were applied. In contrast in 2011, most King Edward and Cara treatments had traces of foliar late blight when first fungicides were applied. At the time the final fungicide application was applied to King Edward, foliar late blight ranged from 0.7 to 94% leaf area affected and 5 out of the 8 trials had >90% foliar late blight. In comparison, for Cara, foliar late blight ranged from 0.5 to 85% and for Sarpo Mira 0.03 to 7.8% leaf area affected. Conditions were generally not favourable for disease development early in the season at the Ayrshire site in 2010. The epidemic was slower to start at the Ceredigion site in the rapid canopy trials in both 2010 and 2011.

Following the logit transformation, which took into account the differences between experiments in the duration of the disease assessment period (and hence differences in the maximum possible AUDPC; equation 3), the regression accounted for 76% of the variation in the logit transformed severity [$P < 0.001$, slope and intercept 95% confidence intervals

201 (0.7144, -1.569) and (0.5341, -2.324)] (Figure 3). T-values [36.01, -15.59] demonstrated that
202 this line was significantly different from the slope of 1 and intercept of 0.

203

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Discussion

The analysis presented here shows that multiplicative survival principles can be applied to derive a simple model to describe the efficacy of host resistance and fungicide dose combinations. To generate predicted AUDPC values and test the ability of the model to predict the joint action of host resistance and fungicide dose, there were two requirements: a fungicide dose response curve (including an 'untreated' control) for a susceptible cultivar (in this case King Edward) plus an 'untreated' control for each test cultivar. Using this method, there was a good relationship between observed AUDPC values and the multiplicative survival model (MSM) predictions.

In a previous study, dose response curves for different host resistance/fungicide dose treatments were compared and differences used to identify the contribution of host resistance. The contribution of host resistance was expressed as the equivalent dose of fungicide required to match the additional disease control provided by a more resistant cultivar (Fry, 1978). An alternative approach, using three dimensional regression, was used to estimate the equivalent fungicide dose equivalent to one point on a 1 to 9 scale (where 9 is most resistant) which defined cultivar resistance (Gans *et al.*, 1995). Such approaches have the disadvantage of requiring dose response curves from all treatments to compare and quantify the benefits of host resistance. This paper demonstrates, for the first time, a method in which the performance of a cultivar in combination with different fungicide doses, could be predicted, without the need to include all the fungicide dose combinations in the experiment and in the absence of a cultivar resistance rating.

MSM models were used originally to determine whether the joint action of mixture components was synergistic. Experimental results are compared with the reference model which represents the joint action predicted from the efficacy of the components. Where the observed severity or AUDPC values were less than, or more than, the predicted values, the

combination may be considered synergistic or antagonistic, respectively (Kosman & Cohen, 1996). For the data presented in this paper, the majority of observed values were below their corresponding predicted values. Cultivar and fungicide combinations therefore performed better than predicted for the majority of host resistance and fungicide dose combinations. Although this is a positive outcome for the value of integrated control, it is important to consider whether this apparent synergy between host resistance and fungicide treatment may in fact have been an artifact of the experimental method.

As described in the methods, mancozeb was applied to all treatments, including 'untreated' plots, once treatment fungicide applications were completed. This over-spray was designed to allow more time for differences in foliar late blight between treatments to develop prior to defoliation. By the time the final treatment fungicides were applied, >90% of the leaf area in untreated King Edward plots, in five out of eight of the experiments, was infected with *P. infestans*. Mancozeb prevents spore germination, but has limited effects on established infections and mycelial growth (Bruck *et al.*, 1981, Kaars Sijpesteijn, 1982). Mancozeb application where > 0.5% of leaf area was affected by *P. infestans* has been shown previously to be insufficient to decrease epidemic growth rate immediately, with a delay of 8 to 10 days before established epidemics were slowed (Fry *et al.*, 1979). It is likely, therefore, that the timing of mancozeb application, relative to epidemic severity and growth rate, differed depending on cultivar and the fungicide dose applied in the experimental treatments. It has been demonstrated previously that the order in which fungicides are applied can also impact on the ability of particular fungicide products to influence the epidemic (Bain & Bardsley, 2009). In the current study, the effect of mancozeb on the epidemic growth rate was likely to be lower for treatments where disease was well established (e.g. on untreated King Edward) compared with other treatments where disease was less established at the time of mancozeb application (e.g. on untreated Sarpo Mira) leading to bias. Such bias could not be

excluded from this analysis, given the absence of a completely fungicide untreated control. Given the apparent effect of oversprays on the epidemic and the potential for bias, it is suggested that such over-sprays should be avoided, or completely untreated controls should be included, for future experiments.

Generating up to date information on the likely efficacy of host resistance and fungicide dose combinations is necessary, particularly when pathogen populations are evolving rapidly towards aggressiveness, virulence or fungicide insensitivity. In Great Britain, the dominance of 13_A2, one of the newer aggressive and more virulent genotypes, resulted in the re-grading of cultivars, including Cara which was used in this experiment, from highly resistant to moderately resistant (Lees *et al.*, 2012). Similarly *P. infestans* genotypes that are less sensitive to and less well controlled by the fungicide fluazinam have been detected in Europe recently (Schepers, 2017). It has been demonstrated previously that the rank order of partial resistance of cultivars exposed to *P. infestans* remains similar with and without fungicide treatment (Bain *et al.*, 2014), therefore using multiplicative survival principles to explore the potential for using integrated control in the way described here should provide a useful guide to the performance of integrated host resistance and fungicide strategies.

Achieving effective control of late blight on potato using decreased fungicide doses on moderately resistant cultivars has been demonstrated previously (Fry, 1975, Clayton & Shattock, 1995, Gans *et al.*, 1995, Bain *et al.*, 2014) and, in some instances, such information has been incorporated into models to guide fungicide applications (Nærstad *et al.*, 2007, Liu *et al.*, 2017). For potato late blight, foliar resistance ratings are calculated for cultivars in many countries (e.g. AHDB, 2017). For the cultivar resistance ratings reported in Europe, 1 to 9 ratings are based on AUDPC values for untreated test cultivars, expressed relative to AUDPC values for one susceptible and one resistant reference cultivar. Cultivars with the

same 1 to 9 resistance rating will therefore have very similar relative mean AUDPC values. Consequently, one resistance rating in the equation should also give a useful indication of the combined efficacy of fungicide doses on all cultivars with the same rating, e.g. there are currently 11 cultivars in Great Britain rated 7 for foliar blight resistance.

The method defined in Equation 2 should be more generally applicable both for potato late blight worldwide and for integrated control in other pathosystems – although further experimental proof is required. The experimental data required to estimate parameters is relatively simple to obtain and analysis is straightforward. Models to describe the joint action of fungicides mixtures, based on MSM principles, have been included as a component in decision support system models for winter wheat disease control (Paveley *et al.*, 2003, Milne *et al.*, 2007) and models of pathogen evolution which consider integrated control (Carolan *et al.*, 2017). MSM principles have proved to be remarkably generalisable in their application, provided the control methods for which joint action is being calculated are reasonably independent. The principles have proved useful for predicting joint action of fungicides, herbicides and insecticides, joint action of separate fungicide treatments in a spray programme (Paveley *et al.*, 2003), joint action of host resistance QTL (Grimmer *et al.*, 2015) and now for the joint action of host resistance and fungicides.

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Figures

Figure 1. Dose response curves (lines) derived from data in the rapid canopy experiments and fitted to the original AUDPC values (points) for King Edward using the equation (1) derived from Paveley *et al.*, 2000. Grey lines are the Ayrshire site and black lines the Ceredigion site. Solid lines are data from 2010 and dashed lines are data from 2011.

Figure 2. Relationship between observed and predicted disease severity for Cara (circles) and Sarpo Mira (triangles) from rapid and stable canopy trials conducted in 2010 and 2011. Black data points identify data points from Ceredigion and white data points from Ayrshire.

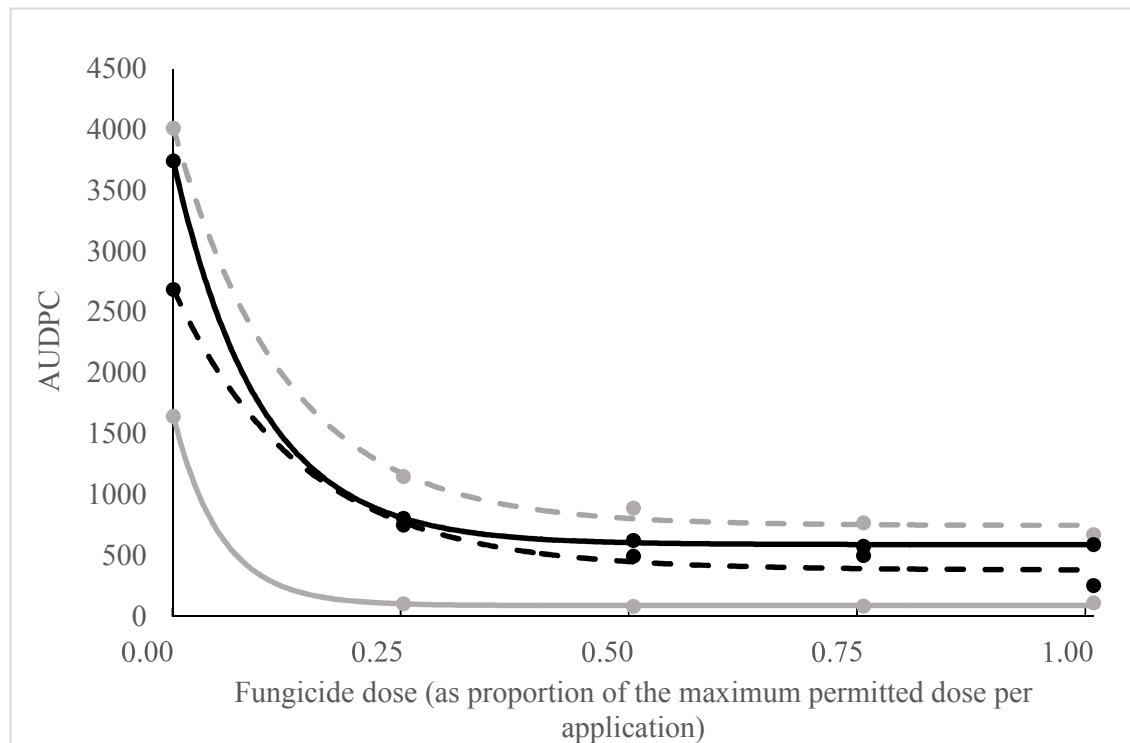
Figure 3. Relationship between logit transformed observed and predicted disease severity for Cara (circles) and Sarpo Mira (triangles) from rapid and stable canopy trials conducted in 2010 and 2011. Slope and intercept 95% confidence intervals [(0.7144, -1.569) and (0.5341, -

395 2.324)]. Solid black line represents the regression and dotted line the 1:1 line. Black data
396 points identify data points from the Ceredigion site and white data points from the Ayrshire
397 site.

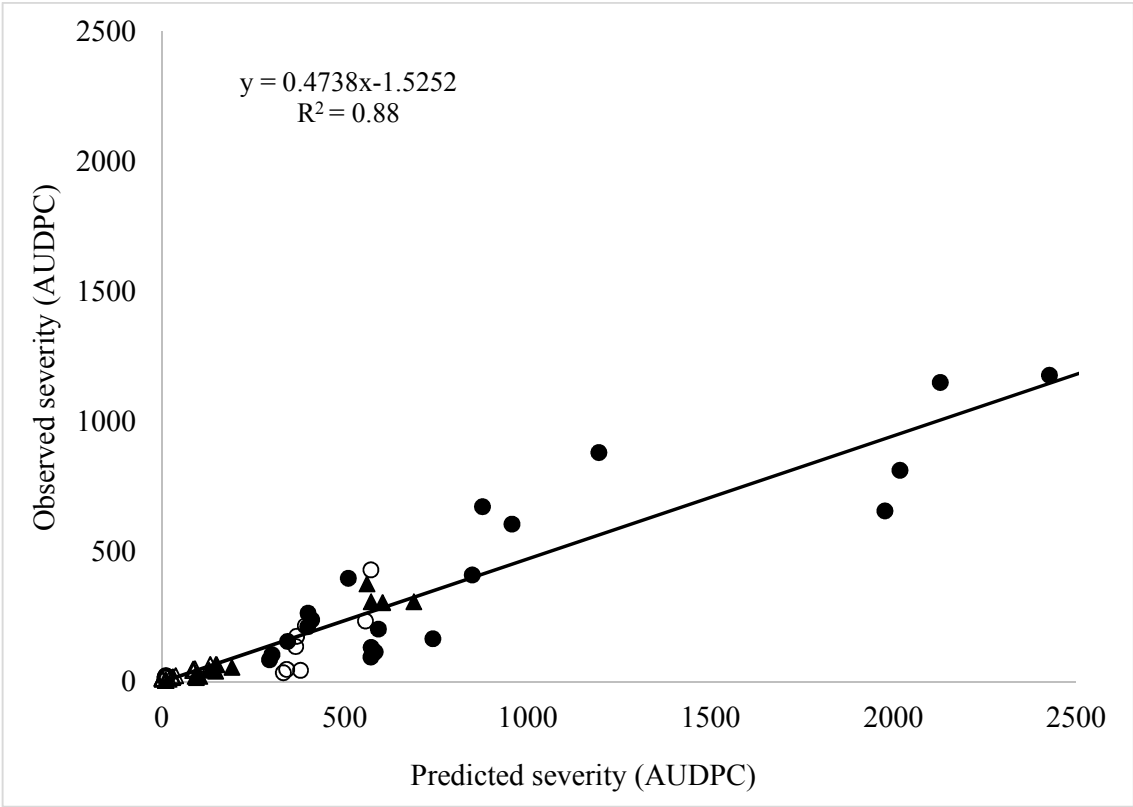
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Figures

Figure 1.



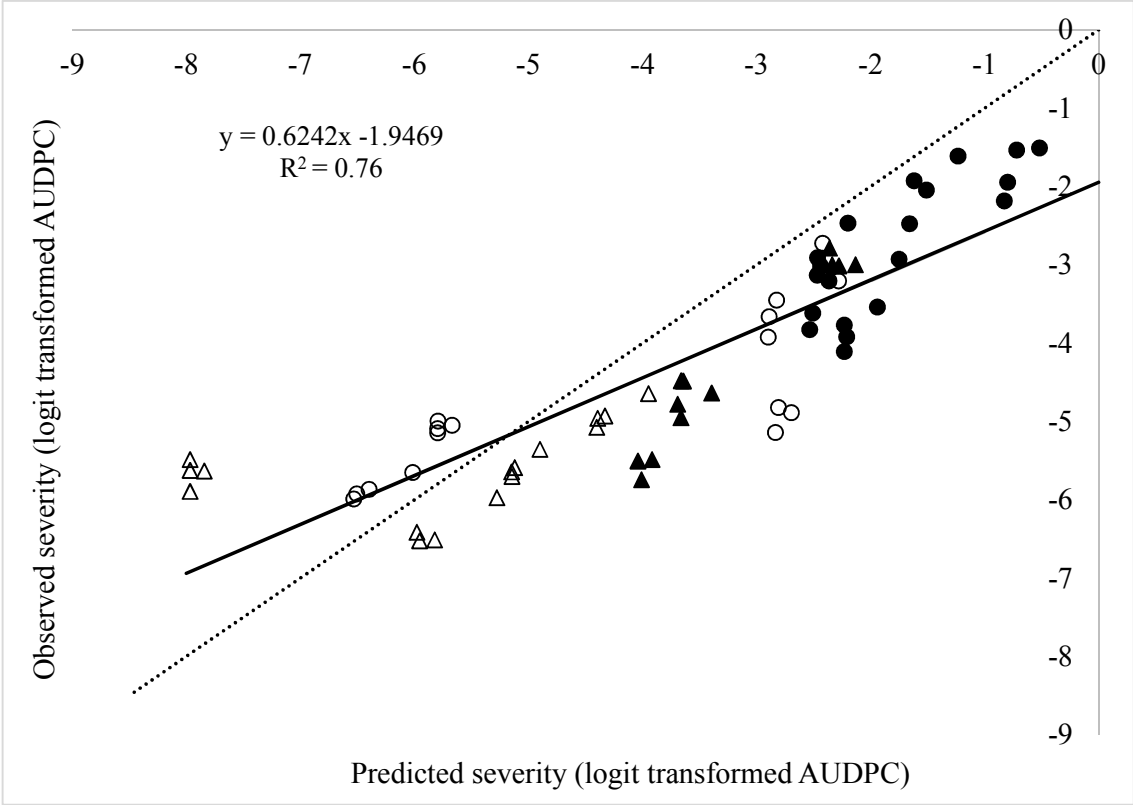
6 Figure 2



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9 Figure 3



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Tables

Table 1. Cultivars, fungicide treatments, spray interval and UK foliar blight resistance ratings for all varieties included in the rapid canopy and stable canopy experiments.

Year	Fungicide doses applied (as percentage of full recommended label rate)	Spray interval	Cultivar (foliar blight resistance rating)*		
2010 and 2011	0, 25%, 50%, 75% and 100%	7 days	King Edward (3)	Cara (5)	Sarpo Mira (7)

*from the Potato Variety Database (AHDB, 2017).

Table 2. *P. infestans* isolate(s) and inoculation dates for each site by year.

Year	Site			
	Ayrshire		Ceredigion	
	inoculation date(s)**	isolates	inoculation date	isolates
2010	15, 19, 28 July 17 August	2009_7654A 07/39, 2009_7654A, 2006_3928A, 2008_6082F	3 July	2009_7654A
2011	8, 13, 18, 28 July 8, 16 August		12 July	2009_7654A

** There were multiple trials at the site, inoculated on different dates.

Table 3. Untreated AUDPC values for King Edward, Cara and Sarpo Mira plus parameter estimates (refer to text for definitions) derived from dose response curves (cv. King Edward/fungicide dose) and R^2 for the fitted dose response curves by site, experiment and year.

Experiment	Site ^a	Year	Untreated AUDPC			Parameter estimates		R^2
			King Edward	Cara	Sarpo Mira	<i>b</i>	<i>k</i>	
Rapid canopy	AYR	2010	1643	196	22	0.94	19.40	1.00
		2011	4008	1957	431	0.81	8.17	1.00
	CER	2010	3741	3042	786	0.81	10.73	1.00
		2011	2686	2058	71	0.86	7.10	0.99
Stable canopy	AYR	2010	3112	1371	36	0.76	6.10	1.00
		2011	2419	1568	116	0.75	9.42	1.00
	CER	2010	3828	3234	918	0.40	3.97	0.96
		2011	2094	1885	204	0.56	4.28	0.98

^aAYR = Ayrshire site, CER = Ceredigion site.

Table 4. Observed and predicted disease severity (as AUDPC) of foliar blight for rapid canopy and stable canopy experiments. Fungicides were applied to rapid canopy experiments early in the season when plants are actively growing and once canopy expansion was complete for stable canopy experiments. Dose is expressed as a proportion of the maximum permitted dose per application and cultivar is expressed as a 1 to 9 ranking, where 1 is most susceptible and 9 is most resistant to *P. infestans*.

Site	Year	Cultivar (resistance rating)	Dose	Trial			
				Rapid Canopy		Stable Canopy	
				Observed	Predicted	Observed	Predicted
Ayrshire	2010	Cara (5)	0.25	23	12	234	557
			0.5	24	11	45	380
			0.75	22	11	48	341
			1.0	21	11	35	333
		Sarlo Mira (7)	0.25	13	1	21	15
			0.5	13	1	17	10
			0.75	10	1	16	9
			1.0	15	1	15	9
Ayrshire	2011	Cara (5)	0.25	431	572	399	510
			0.5	216	392	239	409
			0.75	176	369	264	400
			1.0	136	366	213	399
		Sarlo Mira (7)	0.25	67	133	24	38
			0.5	50	91	19	30
			0.75	49	86	17	30
			1.0	43	85	18	30
Ceredigion	2010	Cara (5)	0.25	167	742	1180	2427
			0.5	115	584	1152	2129
			0.75	133	573	814	2018
			1.0	96	572	658	1977
		Sarlo Mira (7)	0.25	57	192	309	689
			0.5	66	151	305	604
			0.75	66	148	309	573
			1.0	41	148	377	561
Ceredigion	2011	Cara (5)	0.25	203	592	883	1195
			0.5	156	344	607	958
			0.75	105	302	674	877
			1.0	85	294	411	849
		Sarlo Mira (7)	0.25	10	20	44	129
			0.5	6	12	22	103
			0.75	6	10	17	95
			1.0	7	10	22	92

Table 5. The percentage leaf area affected by foliar late blight (%) in untreated varieties at the first (First) or within 7 days of the last (Last) treatment application and the number of test fungicides applied to each trial.

Experi- ment	Site ^a	Year	Percentage of leaf area affected by foliar blight (%)						No. of treatment applications	No. of mancozeb applications
			King Edward		Cara		Sarpo Mira			
			First	Last	First	Last	First	Last		
Rapid canopy	AYR	2010	0.0	0.7	0.0	0.5	0.0	0.3	4	6
		2011	5.3	100.0	1.3	47.5	0.5	7.3	5	4
	CER	2010	0.0	17.5	0.0	22.0	0.0	0.3	4	8
		2011	0.0	9.0	0.0	0.7	0.0	0.0*	4	4
Stable canopy	AYR	2010	0.2	95.0	0.1	33.0	0.0	1.0	6	2
		2011	0.1	97.0	0.0*	60.0	0.0	4.3	5	2
	CER	2010	0.0	90.0	0.0	62.5	0.0	7.8	4	5
		2011	0.1	93.8	0.1	85.0	0.0	7.5	4	2

^aAYR = Ayrshire site, CER = Ceredigion site. * = 0.03

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